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CONFERENCE NUMBER — II

A few of the papers presented at the scientific sessions of the 16th Madras State Medical Conference, Vellore on the 22nd December 1961 are published in this issue as the Conference Number — Part II. Three of the available papers on the "Symposium on Precordial Pain" and a paper delivered at the seminar on "Medical Aid in Rural Areas" find their place in this issue.

A good number of original contributions received by the Editor during the last few months are awaiting publication in our forthcoming issues.

It is intended to select a particular topic each month for our 'Abstracts and Excerpts'. 'Amoebiasis' has been chosen as the subject for this month. Abstracts from different sources on a single subject will, it is hoped, be more useful to our readers.

— *Managing Editor.*

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PRECORDIAL PAIN — CAUSATION *

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Pain, it has been said, is one of Nature's earliest signs of morbidity. Few will deny that it stands preeminent among all the sensory experiences by which man judges the existence of disease within himself. There are relatively few maladies that do not have their painful phases. To cope efficiently with the problem of pain requires a sound knowledge of the sensory supply of many viscera and a familiarity with the typical symptoms of many diseases.

The problem of precordial pain is particularly important, since this in the lay mind is always related to the heart.

In strict anatomical terms, the precordium is the area overlying the heart and is limited by 4 points: (1) the apex situated 9 cm from the midsternal line, (2) the 7th right sterno-costal articulation, (3) the 3rd right costal cartilage and (4) the 2nd left costal cartilage 2·5 cm from the sternum.

In practical terms, however, the precordium is more vague, being the area in and around the sternum and extending on the left side to around the nipple and like the definition is a vague and variable area.

The problem in its different facets will be discussed today by different speakers. I shall confine myself to the causation of precordial pain.

There are as many or more causes of precordial pain as the structures that lie behind and around the precordial area. It will be difficult to catalogue all the causes of precordial pain. I shall only attempt to mention a few of the important and commoner ones.

CAUSES OF PRECORDIAL PAIN:

I Pain arising from the heart

II Other intrathoracic viscera

III Pain arising from the chest wall
- (1) Primary
- (2) Secondary

* State Annual Medical Conference — Vellore, 1961.

IV Sub-diaphragmatic causes

V Mainly functional disorders.

Pain arising from the heart: This is due to 2 main causes — (1) Myocardial hypoxia and (2) pericarditis.

The recognition and differentiation of cardiac pain is important, because of its potential serious consequences. Pain from the heart is ischaemic in origin and is always caused by hopoxia or an insufficient supply of oxygen to the heart.

Factors governing the oxygen supply to the heart:

I Calibre of the coronary arteries

II Blood pressure and cardiac output

III Heart rate

IV Haemoglobin content per unit of blood

V Oxygen saturation of arterial blood.

The oxygen supply to the heart is determined mainly by: (1) the calibre of the coronary arteries, (2) the blood pressure - the flow being generally proportional to the mean pressure, (3) the heart rate and (4) the haemoglobin content per unit of blood.

The oxygen needs of the heart are governed primarily by the amount of work to be performed. The increased demand may be temporary as in exercise, after eating or when the heart rate is elevated. The increased demand may also be permanent as in cases of aortic stenosis or hypertension due to cardiac hypertrophy.

It is when there is a diminished oxygen supply or an increased demand that we get cardiac pain. Clinically this is most commonly associated with coronary artery disease due to arteriosclerosis. The cardiac hypertrophy and low cardiac output present in many conditions like aortic stenosis and incompetence as also any severe congestive cardiac failure can produce cardiac pain. This is particularly true of aortic valvular disease. In cases of aortic stenosis angina of effort is present in around 50% of cases.

Reflex coronary vasoconstriction may also play an important role in some instances. This is probably the mechanism responsible for angina being more easily provoked or induced by digestive disturbances. Thus relative well being and improved exercise tolerance on an empty stomach is a very common symptom in patients with angina pectoris.

A factor which has received relatively little attention and which may be of importance in certain cases is the delay in reflex coronary dilatation. This may account for the rare instances in which pain appears on effort and then disappears, though the exercise be continued at the same rate.

Pericarditis: For a long time it was debated whether the pericardium was capable of giving rise to pain. The studies of Capps indicate that the only sensitive region is the lower part of the external surface of the parietal layer. The pain associated with inflammation of the remaining part of the pericardium actually arises in the adjacent pleura. This fact probably explains why non-infectious pericarditis as in uraemia is painless while infectious pericarditis being nearly always accompanied by pleuritis is usually associated with pain of a typical pleural character. Tension of the pericardial surface is also probably important. Thus after

opening the pericardium, as in cases of mitral valvotomy even with an extensive pericardial rub, retrosternal catching pain is rarely present. Here the pericardium is only loosely sutured.

Clinically in this country pericardial pain is most commonly associated with tuberculous pericarditis and rarely with a respiratory infection as in benign idiopathic pericarditis or in septicaemia.

Aorta and large vessels: Dissecting aneurysms can produce pain indistinguishable from that of myocardial infarction. They are most commonly due to medial necrosis. Their frequency is difficult to assess and are probably underdiagnosed. The incidence would no doubt be in some measure related to the enthusiasm of the doctor and also a careful searching examination.

It is questionable whether syphilitic aortitis uncomplicated by aortic insufficiency, aneurysm or involvement of the coronary ostia gives rise to pain. The pain with aneurysms is almost always due to pressure on the adjacent bones, nerves or ganglia. Occasionally the pain in an aneurysm of the aortic arch appears to be due to irritation of the afferent nerve fibres in the outer coat of the aneurysm. Operative removal of the upper thoracic autonomic ganglia has completely relieved the pain. Brindley and Stembridge have reported on 369 cases of aortic aneurysms. Of these only 68 had a pulsating mass, but 172 complained of pain.

The pulmonary artery: Patients with severe pulmonary hypertension, as in mitral stenosis or septal defects, may complain of pain indistinguishable from that of angina pectoris. This is related to a low cardiac output.

One does, however, also find patients with a large pulmonary artery without pulmonary hypertension or a low cardiac output complaining of dull precordial pain. This pain is probably related to stretching of the afferent receptors in the vessel wall.

Pulmonary embolism, which is fortunately infrequent, can also give rise to precordial pain. This may be the result of a large embolus producing sudden lowering of the cardiac output, and pain indistinguishable from myocardial infarction, or smaller, repeated emboli as in cases of chronic congestive cardiac failure, giving rise to small infarcts, overlying pleuritis and consequent pleural pain.

The mediastinum: In addition to insidious processes like lymphomas, reticulosis or secondary deposits, the mediastinum is at times the seat of an urgent acute process like mediastinal emphysema. Air can reach the mediastinum from many sources. Thus it may: (1) track along the fascial planes of the neck, (2) through a perforation of the trachea, bronchus or oesophagus, (3) from the retro-peritoneal space or (4) from the interstitial tissues of the lung. Interstitial emphysema is commonly caused either by trauma or increased intra pulmonary pressure as occurs when a person strains with the glottis closed. Occasionally it may be due to a spontaneous rupture of the alveoli.

The pain in this condition may come on suddenly. It is due to the irritation of mediastinal structures and also due to pleural irritation. I have not been able to find out from the small series in this hospital or from reports whether the pain is more severe in patients with a great pressure build-up and signs of obstruction.

The respiratory syndromes: The only structures that are sensitive to painful stimuli are the trachea, main bronchi and the parietal pleura.

The unpleasant soreness felt retrosternally in cases of upper respiratory infection is due to the incidental involvement of the trachea.

Any pulmonary process to invoke pain must extend to the pleura or otherwise reach nerve elements. Thus in malignancy pain is an indication of pleural involvement or infiltrations of subjacent nerves.

However, more commonly we have other self limited conditions like pleurisy or pneumonia giving rise to a sharp localised pain aggravated by procedures like taking a deep breath or coughing or in other words any manouevre which tends to irritate or distend the involved pleural surfaces. Studies using curare have shown that spasm of the muscles of the chest wall may also be an important factor in the production of pleural pain.

Epidemic pleurodynia is a relatively rare viral disease which causes pleural pain.

In pneumothorax the pain which is pleural in origin is of a relatively short duration, as the pleural surfaces are soon separated by air.

The oesophageal syndrome: This is being increasingly recognized as a cause of precordial pain. Oesophageal tension appears to be the primary stimulus for the production of pain. The pain or discomfort is thus aggravated by swallowing. Most of these cases are due to a neuromuscular imbalance giving rise to spasm of the lower end of the oesophagus. They are frequently relieved by the use of atropine.

Trapping of an air bubble in the oesophagus, carcinoma, diverticula and peptic ulceration can also give rise to precordial pain.

The pain in hiatus hernia is due to peptic ulceration. The dull ache that these patients complain of on stoopping is related to the increased volume of the thoracic viscera, occasioned by the migration of the stomach. An important additional factor is reflex spasm of the oesophagus.

The chest wall syndromes: Two causes may be recognized: (1) Primary disease of the skeletal tissues. (2) Secondary disorders initiated by visceral disease as in coronary disease.

Tietze's disease affects the costal cartilages particularly of the 2nd rib. There may be visible hypertrophy and tumefaction of the cartilages.

It is important to remember that myeloma, leukaemia and metastatic carcinoma can also produce pain and local tenderness.

Pain arising from the spine or the shoulder girdle or the brachial plexus due to compression is frequently referred to the pectoral region and also the precordium.

One also has relatively ill-defined entities like fibrositis and fascitis with localized tenderness giving rise to precordial pain.

The most confusing conditions, however, are secondary changes in the skeletal tissues due to reflex changes in the muscle tone. The underlying condition is often coronary artery disease. The anterior chest wall syndrome is particularly common and has been well documented by

Prinzmetal *et al.* It occurs most commonly after myocardial infarction. There is tenderness in and around the sternum.

Increased muscular tension due to emotional disease is also believed to give rise to precordial pain.

The sub-diaphragmatic syndromes: There is no doubt that biliary, gastric, pancreatic and splenic diseases can on occasion present with precordial pain and it is important to remember this. I am, however, referring now to the two entities caused by an increased amount of air — (1) in the stomach and (2) the loculation of air in the splenic flexure - the splenic flexure syndrome.

Some degree of air - swallowing is inevitable in the ordinary commerce of the dining table. The first degree of abnormality is reached when the amount of air swallowed obtrudes itself on the notice of the swallower, the second when this is communicated to the bystanders by a series of noisy eructations. Even this second degree of abnormality passes muster as the current coin for appreciation of a good meal in this country.

The importance of aerophagy is its frequency as a cause of precordial pain. The habit may have had a humble origin in excessive salivation or oropharyngeal inflammation. By the time the patient comes to the physician it is usually an ineveterate habit. The pain has been reproduced by introducing air into the stomach. There is considerable evidence that swallowed air constitutes 70% of abdominal "gas".

Not all swallowed air is eructated. It passes through the intestine and during passage may be trapped in the splenic flexure, giving rise to distension and spasm. This is the splenic flexure syndrome and the symptom in nearly 50% of cases may be precordial pain which is relieved by passing wind or faeces and which can be reproduced by introducing air into the splenic flexure.

It is important, however, to remember that these conditions can give rise to reflex coronary vaso-constriction in some patients. Thus it is not infrequent to find true angina pectoris relieved by belching.

THE VICIOUS CYCLE:

Coronary insufficiency ⟶ Angina pectoris
↑ ↓
Reflex coronary vasoconstriction Anxiety
↑ ↓
Increased gastric distension ⟵ Aerophagia

Thus there are innumerable causes for precordial pain. However, in many patients a careful interrogation, thorough examination and detailed investigations often reveal no structural basis for the pain. Closer and more sympathetic questioning will disclose that worry, fear and other emotional states may have aggravated relatively minor aches.

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PRAECORDIAL PAIN — ANATOMICAL AND PHYSIOLOGICAL BASIS

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Man has been always concerned with pain and his earliest records indicate that this attention was invariably voiced in prayers to his Gods. The nature and range of sensations covered by the word "pain" elude precise definition. Aristotle equated pain with unpleasantness whether arising from outside the body or within the soul. Pain has been described by Sherrington as the "physical adjunct of an imperative protective reflex". The evolutionary character of pain as a protective mechanism is indicated by the distribution of pain receptors — for example, the hollow viscera are provided with pain receptors that are fewer in number than those found in the skin.

Most neurophysiologists believe that in the matter of basic perception of pain, there are specific pain receptors-simple, unencapsulated, naked endings – rather than that pain is the result of over stimulation of thermal, tactile or other sensory receptors.

The first sensory neurone is the path by which the sensory impulses from the periphery reach the C. N. S. The cell bodies of the first sensory neurone are situated in the spinal posterior root ganglia and the corresponding sensory ganglia of the cranial nerves. They are bipolar cells, one process being distributed to the periphery and the other entering the spinal cord of the brain stem. The nerve fibres concerned with the appreciation of pain appear to be devoid of end organs and to terminate as free nerve endings. Impulses set up in the peripheral nerves by painful stimulation pass centrally over small myelinated fibres as well as medium sized myelinated ones. The larger fibres are thought to account for the first or pricking pain response. The burning pain response travels over the fine and, therefore, the more slowly conducting fibres.

From all stations in the spinal cord, the second sensory neurone travels across the anterior commissure to the opposite lateral spinothalamic tract and through the brain stem to the postero - lateral and postero-medial nucleii of the thalamus. In the thalamus, third order neurones relay these impulses to the post central cortex as well as to the other thalamic nucleii and to the hypothalamus. Awareness of pain appears to take place at the thalamic level, but the cortex seems to be required for localisation and recognition of the quality and degree of the offending stimulus.

The dual nature of the pain response has been mentioned—the first pricking response and the more slowly developing response. The perception of pain is a physiologic process which involves receptors, conductors and higher perceptive mechanisms. The reaction to pain is a complex psychologic and physiologic response involving the highest cognitive and integrative cerebral mechanisms.

Three types of "pain" are recognised.

Superficial pain intracted by any noxious stimulus which is accurately localised, *deep pain*, of a dull aching, diffuse character and *referred pain.*

Referred pain: Mackenzie, Ross, Head and others have done considerable work in elucidating this problem. The thoracic and abdominal viscera are insensitive to the several types of stimuli which readily arouse sensations in the skin and more superficial tissues of the body. The effects of the different varieties of stimulus - thermal, chemical, tactile and pain have been investigated by a number of workers, including Ross, Head, Mackenzie, Hurst and Carlson. Sensations of pain cannot be elicited from the viscus by the normal means. The intestines, the liver, the stomach or the heart may be cut, burned or pinched without arousing immediate sensation.

The whole subject of pain arising from the viscus and referred from the viscera is still of a highly controversial nature.

Conception of referred pain according to Ross and Mackenzie: When a viscus is diseased, pain and tenderness is felt in the tissues approximately overlying it (abdominal or chest wall), or in some other part quite remote from it. It will be recalled that a given spinal segment supplies a visceral area with autonomic fibres and a well delineated area of skin (dermatome) with somatic nerve.

In the young mammalian embryo, and in certain adult lower forms, e. g. fish, the body is demarcated into a regular series of transverse segments or metameres. The muscles (myotomes), skin (dermatomes) and viscera of each of these primitive blocks eventually receive innervation from the nerve roots of a corresponding spinal segment. The anterior root of each spinal nerve supplies motor fibres to the respective myotome, and autonomic fibres to the viscera and skin; the posterior root supplies sensory fibres to the corresponding dermatome as well as to the muscles and viscera. As a result of the outgrowth of the embryonic limbs, the orderly arrangement of the matameres from before backwards becomes altered.

The two types of structures linked in this way though the cord in embryonic life may be some distance apart, (e. g. diaphragm and shoulder) or be more closely related, e. g. area of abdominal wall and an underlying abdominal viscus. Mackenzie believed that afferent autonomic impulses arising in a diseased organ, though by themselves incapable of arousing any sensation, would upon entering the cord, set up an "irritable focus" and result in stimulation of the corresponding cutaneous nerves and muscle segments. Thus localised rigidity of muscle with cutaneous hyperalgesia and pain occur. Pain is thus referred from the viscus to the skin and muscle segments. All visceral pain is thus "referred" and the actual pain in the viscus is dull, poorly localised and has a sickening quality attached to it.

Physiologic mechanism of pain: The concept that chemicals liberated at the site of injury provoke pain has been supported experimentally by Rewis who found a 'P' factor of pain producing substance liberating at the nerve terminals. Keele and others have demonstrated tiny amounts of three identified substances found in tissues provoke pain when applied to the area exposed after

removal of blistered skin. Both acetylcholine and histamine and 5 HT (serotonin) cause pain. The two later substances have already been associated with injured tissue and serotonin with platelet breakdown.

The Keele group has found an additional pain producing substance (PPS). PPS is found to be different from serotonin and histamine and to resemble the polypeptide bradykinin more closely than any other substance yet tested.

Praecordial pain: The anatomical boundaries of the praecordium have been defined by Dr. George Cherian already.

Pain in the region of the præcordium may arise directly from the skin, subcutaneous tissues and musculoskeletal structures such as the intercostal muscles, ribs and sternum which receive their sensory innervation mainly from neurons located in the dorsal root ganglia of the 2nd to the 6th thoracic nerves. It may also be referred to the praecordium from the thoracic viscera—the heart, the lungs the diaphragm and the œsophagus and from the lining membranes such as the pleura and pericardium.

The heart: The heart and great vessels are supplied by afferent fibres of the vagus nerves and by the cardiac nerves arising from the paravertebral sympathetic trunk. These arise from the upper 5 or 6 thoracic dorsal root ganglia and pass through the thoracic and cervical cardiac nerves. Pain from the coronary vesssels appears to pass over the thoracic cardiac branches of the paravertebral trunk, not over the vagus nerves.

The lungs and its passages: The lungs, trachea, bronchi and smaller air passages are supplied by afferent fibres arising in the vagus nerves. Sensory fibres arising in the upper 6 thoracic roots also supply certain of these structures. Parenchyma of the *lung, smaller bronchi* and the *visceral pleura* are not sensitive to pain. The *trachea and major bronchi* are sensitive to painful stimulation, such afferent impulses appear to pass over the vagus nerves as well as over the thoracic visceral rami of the paravertebral sympathetic trunk.

The oesophagus: The upper thoracic portion of the osophagus is supplied by the 5th, 6th and 7th thoracic roots by direct visceral rami for the sympathetic trunk and also from cervical segments and the lower portion of the oesophagus by the great splanchnic nerves.

Pain may also be referred to the praecordium along the intercostal nerves in conditions such as neuralgias, herpes, nerve root irritation due to spinal cord tumours, etc.

Pain may thus be felt in the praecordium from various causes.

The pericardium and pleura: Whereas visceral pleura is insensitive to pain, the parietal pleura (which lines the inner surface of the chest wall, the upper surface of the diaphragm, the sides of the pericardium and superior mediastinum) receives its innervation from the lower 2nd to 10th thoracic nerves. Pain may thus be referred to the praecordial region.

Whereas the serous pericardium (both visceral and parietal layers) is insensitive to pain, the fibrous pericardium is supplied by the phrenic nerve - C4 and also additional sensory fibres from the upper thoracic segments (D2-D8) reach the pericardium

via the thoracic visceral branches of the paravertebral sympathetic trunk and thence through the cardiac plexuses.

The diaphragm: The anterior and central portions of the pleural surface of the diaphragm are supplied by the phrenic nerves (4th cervical) and partly from the 3rd and 5th cervical roots. The lateral and posterior third of the diaphragm is innervated by the lower 6 thoracic nerves. Involvement or irritation of this portion of the diaphragm may result in lower præcordial pain.

An example of referred pain: In the case of cardiac pain, pain is not felt in the heart, but it is referred to:

(1) Sternum and outer part of left chest (T_2 and T_3)

(2) Down the inside of the arm (C_8 and T_1)

(3) Up the neck to the angle of the jaws to both shoulders and back (C_3 and C_4)

(4) Epigastrium

True cardiac pain is felt in the midline behind the sternum. No local pain is felt in the viscus. A sense of constriction of "angor animi" owing to a visceromotor reflex (due to a reflex spasm of the intercostal muscles which prevents respiratory movements of the chest) is felt.

The cardiac pain apart from its characteristic sites of reference has a diffuse irradiation. This is due to the fact that the sympathetic nerve cells having arrived at the trunk ganglia pass up and down the sympathetic trunk to synapse with nests of ganglion cells at a higher or a lower level and thus form one preganglionic neuron. This explains the diffuse character of cardiac pain and also how pain from the abdominal viscera may occasionally be referred to the præcordium.

Since all viscus are supplied by both sympathetic and parasympathetic, there are 2 areas of referred pain. The corresponding area on the trigeminal distribution in the case of cardiac pain extends on to the forehead and neck and around the eyes. This is thus a rare site of cardiac referred pain.

A knowledge of the pain patterns manifested by individual diseases is the key to the differential diagnosis of pain occuring in or referred to the præcordial region. There is thus no alternative to skilful interrogation of the patient.

PRECORDIAL PAIN — SYMPTOMATOLOGY

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Introduction: The symptoms and signs of chest pain depend on the site of origin and nature of the underlying disease process, whether organic or psychogenic. One has to analyse the location, quality, duration, behaviour pattern, precipitating and alleviating factors. A good knowledge of the common cause of precordial pain is essential and has been already discussed by Dr. Cherian. It has to be remembered that a clinical examination alone may not always give the diagnosis but a detailed history and an intelligent physical examination are the basic requisites in the diagnosis. If pain is the dominant sensory experience in human disease, the correct diagnosis of pain is the proving ground of a physician's medical acumen.

Acute Precordial Pain: *Causes:*

(1) Coronary artery disease
(2) Spontaneous pneumothorax
(3) Pericarditis
(4) Neuritis
(5) Acute respiratory diseases
(6) Pulmonary embolism and infarction
(7) Abdominal catastrophies
(8) Gas pain
(9) Functional.

This is not a complete list, but this serves as a good working table.

Angina pectoris: Anginal pain is the most important and vital cause of precordial pain and a thorough understanding of its nature is essential to diagnose and perhaps, what is more important, when not to diagnose angina.

Site: The pain is central or retrosternal and radiates to the left upper limb, sides of the neck and jaw, across and round the chest bilaterally and occasionally through the back. It is not in the inframammary area though it may be more left pectoral than sternal. Though centrifugal spread is the rule, centripetal spread may occur, with pain starting in the wrist, then on the face and later the chest or may be confined to one of the points of radiation.

Character: Pain in angina is one of constriction, squeezing, pressing or crushing, and occasionally burning, stinging, numbing or sometimes presenting as discomfort rather than actual pain. It is never stabbing, sharp shooting, or throbbing. It is constant while it lasts. The attacks are usually measured in minutes usually two-three minutes. They are not momentary nor last for long hours. The pain is usually produced by any effort that increases the damand for coronary circulation, particularly climbing up or against a wind, running, sudden change of temperature, a large meal, unaccustomed exertion, emotional excitement, anger, fear or apprehension. In severe cases pain occurs on stooping when the patient first goes to bed or on waking up (angina decubitus). Pain that occurs after exertion but not during it and is influenced by lying in some particular position, is not angina. The pain is invariably relieved by rest or glyceryl trinitrate. It must be stressed that certain associated symptoms like dyspnoea, dizziness, faintness, flushing, sweating and

weakness, are not very important. Although they may be provoked by an anginal attack, they are in no way characteristic. There are no typical physical findings associated with angina.

Acute coronary insufficiency: The picture is that of a major episode otherwise resembling an attack of angina of effort. It is the acute counterpart of the chronic process. It is usually acute in onset and unrelated to exertion occuring especially after a meal or walk or excitement. He may wake up at nights with severe sternal pain which does not differ from angina in site or radiation, but is more severe and lasts longer.

There may be associated restlessness and limitation of exercise tolerance, but the pain is still relieved by trinitrate. There is no fever, leucocytosis, raised ESR or change in the B. P. There are still no obvious physical findings except occasionally a gallop rhythm.

Cardiac infarction: The onset is usually sudden though premonitory symptom in the form of an anginal attack may occur in the previous week or so. The attack often occurs without warning or precipitatory causes, started off by pain exactly similar in site, radiation and quality to angina. It is usually more severe, lasts hours instead of minutes, with associated symptoms like collapse, sweating, vomiting, faintness, breathlessness and a feeling of impending disaster. Occasionally there is no pain but syncope or suffocation with paroxysmal dyspnoea or acute pulmonary oedema dominates the clinical picture. Painless infarction does occur, though not as commonly as thought to be, and there are usually physical findings. At the onset he is pale, cold, with signs of peripheral circulatory failure. There may be orthopnoeic cyanosis or collapse. The B. P. falls initially in some cases, but may be elevated in others. It may drop later and remain low for days and occasionally for weeks gradually climbing back to normal in those who survive. The heart sounds are often weak with diastolic or presystolic gallop rhythm. Transient friction rub is heard in 10% of the cases. Arrythmias are not uncommon. There may be a low grade fever. The pain may disappear to leave a sternal soreness which may persist for many days. Frequent attacks of pain following each other ending in death is not uncommon.

Innocent left infra - mammary pain: It is one of the commonest causes simulating ischaemic heart disease. This pain is a long lasting dull ache accentuated from time to time. Situated well to the left, it has no relation to effort and may prevent the patient from lying on his left side. There is often local tenderness.

Pericarditis: Pain may occur in chronic and acute pericarditis. The pain may be precordial or pleural in type, is often dull and aching or sharp, stabbing, cutting pain lasting for hours or days instead of minutes. There is often an association with respiration. The pain may be accentuated by rotating the trunk or by lying on the left side. There is almost always a rub, pericardial or pleuro-pericardial. Fever and tachycardia and general malaise are often found. When effusion occurs, usually the pain disappears. In those who have had a heart operation even after months or years, pain indistinguishable from acute pericarditis occurs associated with fever, malaise and a pericardial rub. This is the

post pericardectomy syndrome. The difficulty arises in those cases where there is local tenderness over the incision. I have seen this happen as late as 18 months after a valvotomy. The cardinal point in the diagnosis is the presence of a rub.

Aortic valvular disease: The pain is the same in character, quality and type as angina and may occur at rest, does not often radiate to the limbs, tends to last longer and is less well relieved by rest and nitrites. The characteristic murmur is heard and evidence of cardiomegaly is more obvious.

In syphilitic aortitis it is more atypical, more nocturnal when lying down and not affected by rest. A diastolic murmur is heard in half of the cases.

Aneurysms of the aorta: These produce pain which may be substernal on either side of the chest depending on the site. There is usually no true relation to exertion. It may be continuous, severe, paroxysmal. When there is erosion of the bone, neuralgia may be present. Depending on the site, signs of decompression on the mediastinal structures will be present.

Dissecting aneurysms: Except for associated hypertension and evidence of arterial occlusion specially in the legs, viscera, brain and face appear like severe infarctions. Few survive.

Respiratory diseases: Pleural pain is maximum in the mammary or axillary region, increased by coughing or deep breathing, is stabbing, tearing, sharp and intermittant and relieved by anything that immobilises the affected side. There is local tenderness and a rub may be heard. The patient may be febrile. In acute lobar pneumonia, as the pain is due to the coincident pleurisy, the symptoms are the same except for fever, tachycardia, localizing signs of consolidation and moist sounds.

Bronchitis: Bronchitis and influenza cause mostly substernal pain which is burning or sore with tightness in the chest, made worse by coughing.

Pneumothorax: Half of the cases have an acute onset with a sharp, tearing or giving away sensation and severe pain in the side of the chest aggravated by breathing. There is dyspnoea, prostration, cyanosis, tachycardia. The affected side does not move well. There is boxy resonance on percussion. The trachea and the apex beat are shifted to the opposite side. The breath sounds are diminished or absent. An x-ray is diagnostic.

Pulmonary embolism and infarction: The onset is sudden with or without severe pain which may be pleuritic or substernal in type and is often bilateral. The dominant symptom is orthopnoea which may be associated with cyanosis. There may be a sensation of oppression. The pain does not radiate. Cough with blood stained sputum is common. There may be evidence of deep venous thrombosis and signs of consolidation in the lungs. A transient rub is present. Massive embolism is ofen difficult to differentiate from cardiac infarction. An arteritis involving the pulmonary artery is a cause of pain resembling intermittent claudication in the chest.

Abdominal catastrophies: The pain is felt in the lower part of the sternum and epigastrium. It is a dull, boring, severe pain, lasting hours, often colicky, radiating to the back and the flanks. A past history of dyspepsia

is often found. There is abdominal tenderness and rigidity or visible peristalsis according to the cause. However, there are times when it is very difficult to come to a correct conclusion. In less catastrophic cases, the pain is related to eating and vomiting and the time relation to the meal is more exact.

Mediastinal syndrome: Acute pain may be due to mediastinal emphysema. Acute mediastinitis or abscess is rare. The pain is behind the sternum, often radiates to the back or upto the shoulder, with superficial tenderness, local signs of fullness or inflammation in the intercostal spaces. Mediastinal crepitations are sometimes heard.

Flatulance: This is a common cause of angina as in splenic flexure syndrome. The pain is usually more upper abdominal and lower thoracic, is dull, distensile and transient. There is often association with meals. The radiation is atypical with no relation to exertion. It may be colicky and aerophagy, belching, water brash and acid eructations are often present. Clinically there may be no signs except vague abdominal tenderness.

Neuritis and neuralgia including herpes zoster provoke pain which has a segmental distribution radiating along the intercostal spaces, is burning, tingling or sore. There is usually hyperaesthesia of the skin, with areas of increased tenderness and later on vesicular eruptions occur in herpes. Acute mastitis, skin infections and adiposa dolorosa have physical findings. Tabes dorsalis and acute transverse myelitis occur with a constricting girdle type of pain.

Psychosomatic: Precordial pain whether chronic or acute is one of the common manifestations of functional disorders. This is the bug-bear of the diagnosticians and over casualness or over emphasis is dangerous. Though functional pain is common, it has to be remembered that a psychoneuritic may also develop coronary thrombosis. The spectrum varies from mild hypochondria to severe psychoneurosis.

A careful history is most important. Functional pain tends to be atypical. It is more often precordical or left mammary than substernal. It frequently lasts for seconds or long hours. The intensity varies and is more often affected by emotional stress. Exertion may relieve pain instead of marking it worse. Rapid heart action, peculiar sensations and so-called weakness are prominent features unlike true angina. Women particularly young ones or near the menopausal age are affected. The pain is often central and radiates all over the body. The attacks are widely spread, stabbing or throbbing pain. The patient often cannot sleep on the left side. Thus it differs from angina in every important requisite-site, character, radiation and duration. The way in which the patient gives the history often provides a clue. A critical cross examination often reveals discrepancies and variations. The pain is rarely associated with any position. Physical findings are inconstant. There may be marked local tenderness and sinus tachycardia. Nitrites do not relieve pain and a local infiltration of novocaine or a placebo may abolish the pain. In the well developed psychoneurotic, the general appearance is one of dejection with a diffident manner, a soft, weak or timid voice

and dull listless facies. In about 30% there is tachycardia, the B. P. may have a raised systolic but normal diastolic pressure, hot flushes and peripheral coldness and cyanosis are sometimes found. Visible sweating is found in over 60%, coarse tremors and asthenic development are not uncommon. In some a heriditary factor is present. The difficult patients are those who have only a mild functional disorder, with none of the obvious stigmata of the psychotic. In these a detailed history particularly whether the story varies on repeated examination is of help. A psychiatatrist's help may be necessary in some patients.

Neuro-circulatory asthenia (Dacosta's syndrome) is only a term to cover all forms of cardiac disturbances associated with a functional disorder. The main symptoms are breathlessness (99%), palpitations (89%), fatigue (38%), left inframammary pain (78%), dizziness 78% and syncope 38%. The cardinal signs are those of a functional disturbance of the respiratory, vasomotor, sundomotor and vascular systems. It must be definitely mentioned that effort syndrome and cardiac neurosis are essentially the same.

Chronic chest pain:

(1) Pericarditis
(2) Mediastinal diseases
(3) Hiatal disorder
(4) Splenic flexure syndrome
(5) Disease of the spine and ribs
(6) Fibro-muscular pain
(7) Functional disorder.

In recent years a lot of importance is being placed upon oesophageal causes and the production of pain which mimics angina. This may be produced by the classical hiatus hernia with ulceration of the oesophagus, or neuromuscular disorders which are being called as hiatal disorders. The pain is usually felt anteriorly under the sternum at a level corresponding to the lesion or may be referred to the back, neck, the jaws and into both arms particularly the inner aspect making it difficult to differentiate from true angina. The cause may be suspected, however, by some relation to food or the aggravation by stooping or lying down and relieved by adopting an erect posture, particularly during and after meals. In well established cases there is often belching, acid eructations and occasionally dysphagia. There are few physical findings except in those cases with a high intra-thoracic stomach, where bowel sounds are heard in the upper chest on auscultation. One has to resort to careful radiological examination for the diagnosis. These disorders are common in the middle age groups.

Chronic mediastinitis and more commonly new growths produce pain varying in nature and distribution according to the site of origin. There are signs of compression of nearby organs like cough, haemoptysis, hoarse voice, venous engorgement, dysphagia, etc.

Spinal caries, rib fractures, cervical rib and spinal injuries are not common. Bone pain tends to be more long lasting and boring in nature. In myalgia and fibrositis, pain is fairly well localised but may radiate and there is usually a trigger point with maximum tenderness. Rheumatism of the chest muscle is a term which is very difficult to define. Muscular pain whether due to a stitch, strain, myalgia or fibrositis produces a syndrome very similar to angina which usually follows excessive exercise or

an exposure to a chill. There are rare forms of chest pain like pulmonary hypertension, periarteritis nodosa and Takayashu's syndrome.

It is true that however well performed a clinical examination may be, a number of patients require further investigations before the cause is found.

DR. T. V. SRINIVASAN, (Tiruchy)
Senior Vice - President
Madras State Branch I. M. A.

MEDICAL AID IN RURAL AREAS *

T. M. KUMARASWAMI, Vellore.

Rural medical relief has been a difficult problem *in our country.* "It has been said that rural people cannot get physicians because there are no hospitals and cannot get hospitals because there are no physicians and may be cannot get either because of lower per capita wealth or income of the rural population.

It is noted that more than 90 per cent of the doctors are concentrated in urban areas with less than 20 per cent population. It is also seen that the proportion of persons per doctor in urban areas is a little over a thousand persons per doctor whereas in rural areas it is forty-five thousand persons per doctor. The great disparity in the provision of personnel, facilities and services for rural areas compared with the urban and the disadvantages under which rural population labours, are well brought out in these figures.

Several solutions to set right this maldistribution have been thought of and schemes such as subsidised medical practitioners, rural dispensaries and rather inadequate health centres, have been tried. Solutions varying from inducement to compulsion have been suggested. What is probably essential is the real understanding of the causes why doctors are not attracted to the rural areas. Apart from economic factors, the others are: lack of ordinary amenities of life, unsatisfactory living and working conditions and more particularly lack of facilities for the satisfactory practice of the profession, viz., facilities for diagnosis and therapy, easy access to specialists; opportunities to gain knowledge and experience, association with other members of the profession and lastly the existence of village factions and interference by outsiders. There is a strange lack of understanding regarding the problems of the profession. It is forgotten that however noble the medical profession is, those who practice it are human beings with human needs and feelings. It is very easy to blame medical men for not moving to the villages, but what are the inducements offered to him in this respect? No other officer belonging to government service is expected to go and settle in a village and why should medical men alone be expected to do so? On the other hand the little that a medical man is able to do for the rural population, inspite of all the handicaps, is not appreciated. A medical man cannot be expected to go to the remotest villages where the authorities themselves have failed to provide the basic needs of everyday life. Even those members of the legislature who have been returned from the rural areas and who are foremost in making appeals to medical men are sticking to towns and cities. To ask the medical man to emulate the example of the christian missionaries is absurd when even a fraction of the benefits enjoyed by them are not given to him. The advisers scarcely take the trouble of ascertaining how well the missions look after the personal

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* A lecture delivered at the Seminar on "Medical Aid in Rural Areas" organised at the 16th Madras State Medical Conference, Vellore on 22—12—'61.

comforts of the doctors even when they work in the remotest jungles. It is futile to appeal to the medical profession to render medical aid in villages with missionary spirit.

Before a medical man could be expected to move to a rural area and settle there, it is essential that all facilities and incentives are created which should include improvement in emoluments, grant of special rural allowance, residential accommodation and assistance for diagnostic help and guidance and facilities for further studies.

Regarding the qualifications of the village doctor, he may not be an expert, but he has to be a physician, surgeon, gynaecologist and so on. In cities things are different. If a doctor is unable to do something, he has experts to fall back upon; in a village it is not so. In fact the village doctor must be more efficient than the city doctor.

In considering as to how the situation could be improved, various solutions have been suggested. The most tempting solution is to compel the doctor to work in rural areas. But I wonder how this can be put through in a democratic society which does not employ totalitarian methods. Practitioners cannot be forced into rural service against their will. They could only be attracted and induced to work in these areas. It is also suggested that prior rural work as being obligatory for medical students to get their degrees. Apart from the harshness of imposing an additional requirement on the medical students who has already to put in 6½ years of study before he can start his career, the villagers will only have the benefit of the inexperienced young medical student who will not be able to help them very much. The time which should be spent by them in gaining real experience in the wards of a hospital is proposed to be spent in areas where they cannot learn anything. So the idea of putting fresh elements in charge of rural areas is of no value.

The next is the proposed introduction of "semi-doctors". With the ever increasing and progressive economy, with the constant race to keep pace with the progress in medical science, it is inconceivable to understand the creation of this cheap and poorly trained doctors. The health of the rural population has been long neglected and they certainly do not deserve a second rate medical attention. There cannot be two different types of medical men attending on two different categories of people. Unless of-course the villagers are intended to be used as 'guinea pigs', the 'semi doctors' will serve no useful purpose. This slipshod method to achieve the aim would be more harmful in the long run although it may appear inevitable in the beginning. I therefore request the authorities to abstain from hastily introducing these "semi doctors".

An immediate, though partial solution of the problem of rural medical relief is by the establishment of mobile units. The district and taluk hospitals may be made the centers of such service. One well equipped mobile unit can bring medical help to villages within a radius of ten miles. I may point out that such mobile units organised by the Christian Medical College Hospital are doing excellent service in villages round about Vellore.

My next suggestion is to let the city and district hospitals be manned as far as possible by honoraries and to utilise the paid staff especially the senior and highly paid medical men in rural areas. This will be a good solution to the problem in the present circumstances when there is no incentive for younger graduates to go to the rural areas.

Another practical proposition for the government to solve the problem is by creating "lend and lease rural dispensaries". The government should build the dispensary with living quarters for the doctor and having built such a unit, they could give it to the doctor on loan re-payable in yearly instalments. Such units are likely to attract young graduates to the rural areas as in course of time the unit will be his property. The government will also realise the capital invested and the scheme is sure to fill up the deficiency of doctors in rural areas.

There is yet another aspect which should be given proper thought and that is creating a separate cadre with higher pay for rural medical work. In the third five year plan it is proposed to increase the number of community development blocks and naturally the number of doctors employed will be large enough to justify the creation of a separate cadre with better emoluments. In this connection, I would like to say that more funds and more attention are needed and even in the third five year plan, out of the total allotment of 300 crores, mfor edical relief (preventive and curative) only 20 crores have been set apart for medical services in community development blocks. How can we achieve any success in augmenting rural medical relief with this meagre amount? The idea of covering the country with a net work of community development blocks is to help the rural population to progress in every direction and more so in the field of agriculture, education and health. The medical activity in the community development programme should be of the type of "total medical care" and to achieve this end more funds and more attention are required.

Another suggestion is to start some sort of health insurance to the villager under a panel system which will induce the doctor to go to the villages as he will have the required patient load. This will cost less to the state and will be a democratic way of meeting the problem.

Lastly, either individual members of the profession on their own or collectivelly through their branch of I. M. A. could go to the nearby rural areas and render medical aid where it is needed. Every one of us may not be a Scudder or a Sommervell with their spirit of service and self-sacrifice, but we can extend our co-operation to the teams organised by the medical associations and institutions like Rotary Clubs, Red Cross Societies and social welfare organisations. Branches of the I. M. A. can even conduct rural hospitals on the lines of missionary hospitals. The government should encourage such voluntary services by giving transport facilities, medical equipment, drugs and other amenities wherever they are required.

So the solution to the problem of rural medical relief is not by making makeshift arrangements or by producing substandard medical men and introducing semi-doctors. While trying to uplift the masses, we are not justified in giving them second rate

medical attention, when in fact they need the services of well qualified and experienced doctors. The problem of rural medical relief is not an isolated one but linked up with the socio-econnomic conditions in the villages and in the absence of the specially created favourable conditions, this problem cannot be solved easily. Considering the various development programmes now going on in various rural areas, this problem, I hope, will find its solution in the near future.

ABSTRACTS AND EXCERPTS

THE CERTAIN DIAGNOSIS OF AMOEBIASIS:

Conventionally the word "amoebiasis" means infection with the protozoal parasite *Entamoeba histolytica.* The infection is cosmopolitan, rather common, and not invariably pathogenic. It is primarily located in the large intestine and is acquired by swallowing cysts of the parasite passed in the stools of another person so infected. Specific diagnosis of intestinal infection with *E. histolytica* is readily made, because some parasites are constantly being voided in the stools. If the stools are loose the parasites appear in their normal parasitic form, the amoeba. The amoebae do not withstand gross environmental change and die and disintegrate within a few (6—8) hours of passage; loose stools must, therefore, be examined for them within this time. But if stools are passed only twice daily, they are in a formed state. This slow carriage of the amoebae to the exterior enables them to prepare for environmental change by encystment of each within a spherical protective envelope. Possibly this encystment is stimulated by the abstraction of water from the intestinal contents in the lower large intestine. At any rate in fluid or loose stools only amoebae are to be found and in formed stools only cysts. The cysts are not parasitic; they occur in no place other than in stools.

Competent microscopical examination of an emulsion of stool preferably repeated on several consecutive days, reveals beyond doubt the presence of *E. histolytica* in all but the most exceptional cases of intestinal amoebiasis, symptomatic or asymptomatic. The diagnosis rests on this. But their recovery and also their differentiation from other protozoa and from the many cells and other confusing structures found in stools require skill. There are techniques for the concentration of cysts from stools; while in theory these should simplify the recovery of the cysts, in practice they do not necessarily do so. Sigmoidoscopy with scraping, or the aspiration of exudate from the surface of the lesion may well yield amoebae. Usually it is considered unnecessary to do a snip biopsy, and the finding of amoebae in any histological section is much more difficult than in smear preparations.

Specific identification of the parasite after its discovery is essential. It is a matter of fact and not of opinion, and the skill to do it comes from special knowledge and regular practice. There are several genera and species of amoebae which may occur in the large intestine of man; some are much commoner than *E. histolytica*. For example, *E. coli*, a frequent harmless commensal, in its amoeboid stage is virtually indistinguishable from the same stage of *E. histolytica*, though its mature cysts are large and they contain 8 and not the 4 nuclei of *E. histolytica*. *Entamoeba* nuclei are morphologically characteristic and their morphology must be determined before an identification of the genus is made. Mobility and motility are not attributes solely of amoebae; they are seen in sundry large phagocytic cells, which also may contain engulfed red cells.

Until the causative parasites are specifically identified the diagnosis in any disease of parasitic causation must remain speculative. A diagnosis made on response to treatment is unsound. This holds good for malaria, kala-azar, trypanosomiasis, and other major protozoal parasitic diseases of man as well as for the helminthic diseases. The proper diagnosis of any of these conditions is of extreme importance to the patient; the specific treatment for each are by no means devoid of risk. It is, therefore, essential to be as sure as possible of the diagnosis before embarking on specific treatment. A justifiable exception may be the early development of hepatic amoebiasis. During this complication the primary parasitic infection of the bowel often falls in intensity, and so few parasites are voided in the stools that they may be difficult to find. In view of the damage wrought by, and the disastrous complications which will result from, a neglected amoebic hepatic infection, immediate specific treatment is justified on a reasonably based clinical diagnosis even if examination of the stools is negative. Prompt treatment may prevent the formation of gross abscess, but if this has already developed, needling of it can confirm its presence. Needling solely for diagnosis is not to be encouraged, but if undertaken and abscess contents are obtained in the aspirate, facilities should be available to complete the evacuation of it. More amoebae are around the margins of the liver lesions than in its centre. Material coughed up by a patient with extension of an amoebic liver abscess into the lung contains recognizable amoebae. The persistent and skilled examiner finds them there with much regularity, as he does in needle aspirates of amoebic cavities in the liver.

The specific treatment of amoebiasis is fairly simple. An actively pathogenic intestinal infection is rapidly controlled by emetine given parenterally, though it is not entirely eradicated. But following this with ten days' oral treatment with emetine bismuth iodide, or possibly one of the proprietary amoebicides, should sterilize this infection. If the infection has extended outside the intestine, the patient should receive parenteral emetine. Chloroquine given orally is an alternative, or an adjuvant to this, though only for hepatic amoebiasis. The primary intestinal infection must be eradicated as the final stage of treatment in every case or the risk remains of clinical relapse or recurrence of complications.

—*British Medical Journal, Feb. 10, 1962—p. 386, Editorial.*

SOME NEWER AMOEBICIDES IN ACUTE AMOEBIC DYSENTERY:

The purpose of this paper is to record the effect of these drugs in acute amoebic dysentery and to compare them with the results of standard therapy in similar patients.

All patients were adult African males complaining of diarrhoea with blood and mucus, in whom rectal ulceration was seen and material from ulcers contained haematophagous trophozoites of *Entamoeba histolytica*. Patients with hepatic amoebiasis or severe dysentery were excluded to avoid risk to life. The period of observation was 27 days in hospital. Endoscopy was done initially and at 5, 10, 15, 20 and 27 days. Stool examinations, (both direct and by zinc flotation when indicated) were done daily while ulceration was present and always at the same intervals as endoscopy.

TABLE

Results of therapy: cumulative result at 27 days; percentage in brackets.

Drug	No of patients	Success	Probable failure	Absolute failure	Parasite failure
ENTAMIDE: 5 gs. daily for 10 days ...	10	4 (40)	3 (30)	2 (20)	1 (10)
ENTAMIDE (enteric coated): 5 gs. daily for 10 days ...	10	1 (10)	4 (40)	5 (50)	—
ENTAMIDE FUROATE: 4 gs. daily for 10 days ...	10	4 (40)	—	6 (60)	—
MEBINOL: 1·8 gs. daily for 10 days ...	31	19 (61)	2 (7)	8 (25)	2 (7)
ENTOBEX: 300 mg. daily for 10 days ...	13	4 (31)	4 (31)	5 (38)	—
HUMATIN: 600 mg. daily for 10 days ...	10	2 (20)	2 (20)	6 (20)	—
HUMATIN: 2·4 gs. daily for 5 days ...	10	6 (60)	—	4 (40)	—
HUMATIN: 3 gs. daily for 10 days ...	10	8 (80)	2 (20)	—	—
TETRACYCLINE: 1 gm. daily for 10 days ...	36	35 (97)	—	1 (3)	—
EMETINE: 65 mg. i.m. daily for 3 days then E. B. I.: 195 mg. daily for 10 days	27	25 (92)	—	1 (4)	1 (4)

ENTAMIDE - Diloxanide

MEBINOL - Chlorphenoxamide

ENTOBEX - 5, 6-quinone-4, 7-phenanthroline

HUMATIN - Paromomycin

Conclusion : With the possible exception of Humatin in high dosage, claims that the preparations tested are adequate treatment for all forms of intestinal amoebiasis should be treated with reserve. In addition, there is indirect evidence that Mebinol, Entobex and Humatin are ineffective in hepatic amoebiasis.

We believe that amoebic dysentery requires treatment by a combination of drugs, one of which should be in preventing hepatic involvement. The value of the drugs discussed when combined with other amoebicides still requires assessment, as they compare favourably with many preparations in current use.

— *Wilmot A. J.*, et-al. *The Amoebiasis Research unit, University of Natal, Durban. Trans. Roy. Soc. Trop. Med & Hyg.* **56**, *85-86 (1962).*

* * * *

AMOEBICIDES:

For all acute clinical manifestations, parenterally given emetine is unfailing and unrivalled as a specific.

For the treatment of hepatic amœbiasis, chloroquine may be substituted for emetine or used to reinforce it.

For the eradication of the infection present in the large intestine, emetine and bismuth iodide is unequalled in the regularity of its achievement; sundry synthetic amœbicides can be substituted for it with less certainty of cure but with freedom from toxic side-effects.

Orally administered antibiotics achieve a lower degree of success for this purpose; all but paromomycin and fumagillins do so by their action on the associated bacterial flora. Paromomycin is under trial for its therapeutic effectiveness as an amœbicide, but judgement on it must await more lengthy and extensive study; fumagillin is unsatisfactory as a specific.

— *A. R. D.* ADAMS, M. D., F.R.C.P., D.T.M., *Liverpool School of Tropical Medicine. Practitioner* **188**, *71–73 (1961).*

ELECTROCARDIOGRAPHIC CHANGES DURING EMETINE TREATMENT OF EGYPTIAN CHILDREN WITH AMOEBIC DYSENTERY :

Thirty-three children two to 12 years old, suffering from acute amoebic dysentery were treated with emetine hydrochloride. The ECG changes were evaluated during and after treatment. Minor and very transient changes were encountered in 36.3 per cent of the patients.

This study shows thaat emetine in the treatment of amoebic dysentery in children in the doses mentioned (1mg per kg body weight per day) is safer than for adults, and that there is nothing to support the general trend of refraining to employ this drug in paediatric practice. It also shows that in children, there is no need for the use of supportive drugs commonly combined with emetine therapy in treating adult patients.

— *Awwaad S. and others, Ain Shams University, Cairo. J. Trop. Med & Hyg.* **64**, *286-287 (1961).*

* * * *

THE TREATMENT OF AMOEBIASIS WITH 'RESOTREN COMPOUND' :

In a period of observation extending from three to four months, 'Resotren Compound', (a mixture containing 75 mg. 'Resotren', 200 mg. chloroquine phosphate as 'Resochin', and 300 mg. of diiodohydroxyquinoline per tablet) was found to possess marked amoebicidal activity *in vivo* for the treatment of acute and chronic amoebic dysentery and amoebic hepatitis.

When given in doses of six tablets per day for the first week followed by three tablets per day the next week, it cured 86 per cent of the cases. The present series included 27 cases of acute amoebic dysentery and 59 cases of chronic amoebic dysentery, the latter group including seven cases of amœbic hepatitis. The 11 cases that did relapse were recorded among the chornic cases.

Side-reactions were recorded in 20 per cent of cases and necessitated reduction of the daily dose to three tablets daily, but the total dose was given, spread out over three weeks, with the same rate of apparent cure.

— *Shafei A. Z., Alexandria University,* U. A. R. *J. Trop. Med & Hyg.* **64**, *282-285 (1961).*

ASSOCIATION NOTES

BRANCH NOTES

Anamallais branch:

1. The monthly clinical meeting was held on 27th January 1962 at Paralai Estate with Dr. John in the chair. 8 members were present. Dr. M. P. Gundappa presented an interesting case report on "Myeloid Leukaemia", followed by a discussion by the members.

2. A clinical meeting was held on 16th February 1962 at Mudis with Dr. John in the chair, 7 members were present. Dr. K. Janardhanan gave a concise talk on "Menopause and its management" followed by a short discussion. Dr. M. James was bid good wishes and farewell on the eve of his departure to Nilgiris.

3. At a clinical meeting held on 30th March 1962 at Mudis with Dr. John in the chair, 7 members were present. Arrangements for the annual meeting day celebrations in April were fully discussed.

Coimbatore branch:

1. The monthly meeting of the Coimbatore District Medical Association was held as a joint meeting with the Palghat District Medical Association at Palghat. 12 members from our branch participated. Dr. T. A. Venkatachala Iyer, president of the Palghat District Medical Association presided and introduced the speaker, Dr. R. Mahadevan, M. S., F. R. C. S., Retired Prof. of Surgery, Madras Medical College and requested him to address the gathering. He gave an interesting lecture on the "Nature of the diseases found in our country contrary to the text book descriptions". He stressed on the need of correct entries in 'case sheets,' so that they will be of immense help for future reference. He requested that the maternity benefit given to the workers may be utilised for family planning purposes after 3 live births.

2. The monthly meeting of the Coimbatore District Medical Association (branch of the I. M. A.) was held on Saturday, the 17th February at 5 p. m. in the premises of the association. 100 members were present. Dr. G. T. Gopalakrishna Naidu, L. M. P., L. P. H., the president presided. A group photo was taken of all the members present. A condolence resolution was passed touching the death of Dr. T. K. Subramaniam, an oldest member and past president of our association was adopted. The president introduced Dr. C. H. Sivaraman, M. B., B. S., M. R. C. P., D. T. M. H., (Edin), Divisional Medical Officer, Southern Railway, Podanur and requested him to deliver his address. Dr. C. H. Sivaraman then addressed the gathering on 'Chronic Respiratory Infections". He spoke on P. T., tropical eosinophilia, sarcoidosis and bronchiectasis. He elaborated on the modern trends of treatment and the necessity to remember sarcoidosis. There was the

usual discussion after the lecture. Dr. P. K. Kalyanaraman and Dr. N. Subramanyan also gave an account of their own experiences of the above diseases especially tropical eosinophilia.

Madurai branch:

The general body and monthly meeting of the Madura Medical Association was held on Saturday, the 24th March 1962, under the presidentship of Dr. K. Ramachandran, M. S., Madurai. Dr. A. Venkoba Rau, M. D., D. P. M., F. I. P. S., Assistant Professor of Psychiatry, Stanley Medical College and Assistant Surgeon, Government Mental Hospital, Madras gave an interesting lecture on " Depression - some aspects and management "

Pudukottah branch:

An ordinary meeting of the association was held on 25—3—1962 at the Town General Hospital, Pudukottai. Dr. C. R. Thiruvengadam, M. B., B. S., Chief Medical Officer presided. Eighteen members and four guests were present. Dr. S. Shanmugam, F. R. C. S., Hony. Asst. Medical Officer, Govt. Erskine Hospital, Madurai addressed the meeting on the subject of " New trends in surgery ". Dr. Bhaskar Rao, M. D., Professor of Obstetrics and Gynaecology Erskine Hospital, Madurai gave a short description of " Puerperal, Sterilization ".

Salem branch:

The monthly meeting of the Salem branch of the Indian Medical Association was held on Saturday, the 21st April 1962 at Foulke's Hall, Salem. Dr. Jayaramachandran presided. Dr. A. Krishnaswamy, M. B., B. S., D. M. R., Superintendent, Erskine Hospital, Madurai spoke on " X-ray Diagnosis of Kidney Diseases ". Dr. K. A. Ramalingam, M. B., B. S., F. R. C. S., Honorary Lecturer in Orthopaedics, Madurai Medical College spoke on " Trends in orthopaedic practice ".

Trichy branch:

1. A monthly meeting of the association was held on Saturday, the 31st March, 1962 in the association premises. 40 members were present. Dr. T. V. Ranganathan, the president presided. Dr. G. C. Anbunatnan, M. B., B. S., M. R. C. P., D. T. M. & H., Hony. Physician, Erskine Hospital and Hony. Lecturer in Therapeutics, Madurai Medical College, Madurai addressed the members on " Emergencies in medicine ".

2. A montkly meeting of the association was held on Saturday, the 7th April, 1962 in the association premises. About 40 members were present. Dr. T. V. Ranganathan, the president presided and introduced the lecturer of the day, Dr. E. Balakrishnan, M. S., D. O., Asst. Professor, Elliot School of Ophthalmology, Madras to the members. The lecturer gave an excellent talk on " The usefulness of ophthalmic signs in general practice " and also showed a number of beautiful colour slides on the subject. There was a lively discussion on the subject and many members took part.

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BUTARIN
INJECTABLE & TABLETS

Sole Distributors: **THEMIS DISTRIBUTORS, 43, Queens Road, BOMBAY-2.**

ADVANCE

CONTROL HYPERTENSION
Successfully with

RALFEN

STANDARDIZED EXTRACT OF RAUWOLFIA SERPENTINA

- Causes marked but gradual fall in blood pressure.
- Lowers both systolic and diastolic pressure.
- Relieves distressing nervous symptoms of hypertension.
- Is a mild hypnotic and produces refreshing sleep.

Supplies :

Liquid Extract : In 28 ml. phials.
Tablet of 1·6 mg. : In bottles of fifty.

★

BENGAL CHEMICAL
CALCUTTA - BOMBAY - KANPUR

TO LET

EPHOMAG

A DEPENDABLE ORAL ANTI-ASTHMATIC

FOR PROMPT RELIEF OF ASTHMA, BRONCHITIS, SPASMODIC COUGHS, etc.

ELIXIR
Contains:
Ephedrine Hcl, Aminophylline, Sod. Iodide, Sod. Benzoas, Caffeine Citras, Belladonna, Lobelia, Adatoda, Glycyrrhiza, Senega, etc.

FORTE TABLETS
Contains
Ephedrine Hcl
Aminophylline, Caffeine Citras, Phenobarbitone
Aluminium Hydroxide
Belladonna &
Calcium
Gluconate

DATA TEMPORE MPL PROSUNT

M.A.C. Margarets Pharma - Labs.

40, SAN THOME HIGH ROAD, MADRAS-28.

Telegrams: MAGMARS
Telephone: 71886.

EP

ACIDOMAG

(TABLETS)

Antacid, Gastric Sedative & Digestive corrective.

FERIMAG 12

Elixir & Tablets
GENERAL TONIC & RESTORATIVE

Indicated in Anaemia, Malnutrition, Convalescence & Weakness after childbirth.

DATA TEMPORE MPL PROSUNT

ELIXIR
Contains:
Liver Extract, Vitamin B 12, Folic Acid, Vitamin B Complex, Niacinamide, Exsiccated Ferrous Sulphate, Sodium Glycerophosphate, Manganese Glycerophosphate, Traces of Copper & Strychnine Hydrochloride.

TABLET
Contains:
Exsiccated Ferrous Sulphate, Vitamin B 12, Folic Acid, Calcium Gluconate, Vitamin D 2, Vitamin C.

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EP